

The NCVDLS REPORT



*Veterinary News and
Information From North
Carolina's Diagnostic
Laboratories*



North Carolina Department of
Agriculture and Consumer Services
Steve Troxler, Commissioner

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Holiday Closings...

September 1, 2014

November 11, 27 and 28, 2014

December 24, 25 and 26, 2014

Please e-mail
NCVDL@ncagr.gov with any
comments and/or suggestions
concerning The NCVDLS
Report
Editor - Dr. David Drum

Message from the Director

It is with much regret that I report the impending retirement of our State Veterinarian and my boss, Dr. David Marshall. His retirement becomes effective on September 1, 2014. Dr. Marshall, besides being a former Director of Laboratories, is currently the NCDA&CS, Veterinary Division Head. His stint as Director of Laboratories provided him with an awareness as to the importance of our services and what is necessary to maintain them. He has also been a great supporter of the NCVDLS as Division Head in terms of approving funds that have improved our facilities, upgraded our equipment, improved our information technology services, and supported continuing educational opportunities for our personnel. We were fortunate to have such a great advocate and wish him a well-deserved retirement.

Karen W. Post DVM, MS



Client Corner

Swine Enteric Coronavirus Disease (SECD)

Porcine Epidemic Diarrhea Virus (PEDv) was first recognized and confirmed in April and May of 2013. The virus spread throughout the Midwest. It was first identified in North Carolina in June 2013 and has quickly spread with greater than 700 cases currently reported in North Carolina.

PEDv is a member of the family Coronaviridae and is clinically similar to transmissible gastroenteritis (TGE) but unrelated. Prior exposure or vaccination to TGE does not provide immunity. The introduction of PEDv into a naïve herd typically results in severe diarrhea, vomiting, high morbidity and variable mortality. Mortality in young pigs up to two weeks of age is routinely 100%.

There is recent evidence of strain differences in PEDv, and a Swine delta Coronavirus (SDCoV) has also been identified. The NCVDLs has PCR tests available for both PED and SDCoV. The PED PCR test detects the variant PEDv strain and the laboratory continues to evaluate testing options for these viruses.

Effective June 5, 2014, the US Secretary of Agriculture by Federal Order requires producers, veterinarians, and veterinary diagnostic laboratories to report new cases of SECD (PEDv and SDCoV) to the appropriate state and/or federal animal health officials.

Please call the Rollins Laboratory regarding testing questions.

- Dr. Richard Mock, Assistant Laboratory Director

Feature Article

Winter and Spring Cases of Cockerbur Toxicosis

Jennifer Haugland, DVM

Herd #1

In February 2014 a herd of mixed breed beef cattle lost two cows and four calves within a three-four day period. The reported clinical signs were acute death, ataxia, ptyalism, and recumbency. Hay was the only source of feed. A 4 month old calf was necropsied at the Rollins Laboratory. The calf was in thin body condition and there was serous atrophy of fat. Additional gross lesions included clear yellow fluid in the abdominal cavity, pale liver, a lack of milk in the abomasum and decreased ingesta in the intestines.

Herd #2

In May of 2014 a dairy farm (Herd #2) lost 10 eight month Holstein heifers from a group of 20 in less than 48 hours. On the first morning one calf was found dead and then three more were found dead a few hours later. During the entire day, no calves appeared sick. On the next morning five more calves were found dead and one calf was moving slowly. This slow moving calf died a few hours later. This group of calves was fed hay and a 5 gallon bucket of grain mixed at the farm twice a day. The grain mix was also

Feature Article continued

being fed to all calves and the lactating cows. The owner reported that the neighbors had thrown watermelon rinds over the fence and that there were more weeds than grass in the pasture. The owner was able to identify some weeds as buttercups and others were possibly young cocklebur plants. Two calves from the first day were necropsied at the Rollins Laboratory. The slow moving calf was necropsied by the field veterinarian and formalin fixed tissues were submitted to Rollins Laboratory. An 11th dead calf from a different field was found dead on Day 3 and it was also necropsied at the Rollins Laboratory.

Necropsy examinations revealed similar gross lesions from all 4 calves and they were clear, yellow peritoneal fluid, marked edema in the wall of the gall bladder and edema around the duodenum and pancreas. Cocklebur burs were found attached to the chin of the 4th calf examined. Samples of ocular fluid from the calves necropsied at the laboratory were negative for nitrate-nitrogen. Abundant corn was found in the rumens and abomasums of the first 2 calves and the pH of the rumen fluid was 5.5 and 6.0. Rumen acidosis was the preliminary diagnosis from the examination of the first 2 calves. However, rumen acidosis was considered much less likely when more calves died the second day and it was confirmed that only 2 five gallon buckets of grain were fed to the entire group. A toxin was then considered more likely.

Diagnosis

Multiple tissues of all necropsied animals from both herds were examined microscopically and the histopathology revealed moderate to severe centrilobular to midzonal necrosis of the liver in all cattle examined from both herds.

Possible causes of zonal necrosis in the liver include hypoxia and toxins. In cattle, toxins that can cause zonal hepatocellular necrosis include toxic plants and chemicals. Plants from the eastern US include cocklebur, cottonseed/ gossypol (also cardiac damage), toxic mushrooms (*Amanita* spp. and *Galerina* spp.), oak buds or acorns (also severe kidney damage) and toxic blue-green algae. Hepatotoxic chemicals include carbon tetrachloride (CCl₄), C1-hydrocarbons, polybrominated biphenyl (PBB fire retardant), carbon disulfide (CS₂), coal tar pitch (clay pigeons, pipe sealer, tar paper), phenol (wood preservative), tannic acid, paraquat/diquat (also lung damage), aflatoxin B (very high levels 3mg/kg), rubratoxin B, ochratoxin A (also kidney damage), oil of pennyroyal, and hexachloroethane. (Large Animal Internal Medicine, Smith B.; 3rd ed. 2002)

In Herd # 1 the owner reported seeing weeds and cockleburs in the hay. In Herd #2, no sources of chemical toxins were found in the pasture examined and the only suspicious plants were buttercup and seedlings that were possibly cocklebur plants. Buttercup plants may cause colic, diarrhea, and nervousness but do not cause liver necrosis. Photographs of the seedlings suspected to be cocklebur were forwarded to the Plant Biology Department at NCSU. The seedlings were identified as common cocklebur (*Xanthium* spp.).

In both herds, the plant, cocklebur (*Xanthium* spp), was considered the most likely cause of death based on the histopathology findings and the presence of either mature plants in hay or the seedlings in pasture.

Cocklebur toxicosis results from ingestion of the dicotyledonary (2-leaf) stage, seedling stage, or seeds of the common cockleburs. **(Figures 1 and 2)** Cocklebur are ubiquitous throughout North America and can be fatal when consumed by livestock via pasture, feed contamination with bur seeds, in hay, or while grazing crop residues.

Feature Article continued

Cases of cocklebur poisoning have been reported from cattle, sheep, swine and poultry. Consumption of as little as 0.75% of body weight of cotyledonary portions can cause death, with clinical signs occurring a few hours post cocklebur plant or seed ingestion. The most commonly reported clinical signs of cocklebur intoxication in pigs and cattle include depression, weakness, anorexia, reluctance to move, opisthotonus, ataxia, hyperexcitability, spasmodic muscular activity, and an unusual gait with erect ears and head held high. Recumbency, paddling of limbs, convulsions followed by coma, and death can occur within hours to days of consumption. Onset of clinical signs is usually delayed in cattle with a functional rumen as compared to non-ruminants. Sudden death with no clinical signs is possible in calves.

(2007 Newsletter from the ADDL at Purdue University <https://www.addl.purdue.edu/newsletters/2007/Summer/CT.html>)



Figure 1 – Cocklebur seedling and cotyledonary stages.

Note the burr between the arrows



Figure 2 – Burr can be found still attached to the root of seedling.

Short Cuts

COMPANION ANIMAL

Exotics

In June 2014, two adult reticulated python snakes were submitted to Rollins Laboratory for necropsy with a history of death followed by a one week history of regurgitation, raspy breathing and development of full body skin rashes.

The female snake measured 14.5 feet in length and weighed 35 kg. No skin lesions were identified. Mouth rot was present, characterized by severe diffuse gingival hyperemia, edema and proliferation with chunky fibrinonecrotic debris adhered to the mucosa. Petechiation and multiple purple to black foci were identified along the glottal mucosa. Multiple white firm round to oval nodules measuring up to 7 mm in diameter with pitted centers containing firm white-yellow debris were distributed along the mucosa of the distal esophagus and throughout the stomach (**Figure 1**). Petechial hemorrhages with white to yellow raised plaques were identified along a half-foot long section of the proximal small intestine.

The male snake measured 8.5 feet in length and weighed 6.5 kg. Random scales were individually red-dened along the body with no evidence of swelling. Similar nodules identified in the first snake were also identified in the proximal esophagus, peri-esophageal connective tissue, and colon of this snake. Petechial hemorrhages with slightly raised yellow/tan nodules measuring up to 3 mm were identified along the mucosa of the distal small intestine. A 2 cm rectal prolapse was identified.

Histologic examination of these snakes revealed eosinophilic intracytoplasmic inclusions in the epithelial cells of multiple organs, mainly the collecting ducts and pelvis of the kidneys (**Figure 2**). There was an erosive to ulcerative, fibrinosuppurative rhinitis, stomatitis and tracheitis, fibrinosuppurative splenitis, and fibrinonecrotic and pyogranulomatous esophagitis, gastroenteritis and colitis (**Figure 3**). Mixed bacteria were present in these lesions. Also identified were random heterophilic and lymphoplasmacytic to pyogranulomatous hepatitis, multifocal pustular dermatitis, multifocal pyogranulomatous interstitial pneumonia with pneumocyte necrosis, pyogranulomatous orchitis, and suppurative endometritis. The intracytoplasmic inclusions were consistent with inclusion body disease in snakes. The numerous inflammatory changes were indicative of a secondary bacterial infection.

Inclusion body disease (IBD) is seen worldwide in snakes that are members of the families Boidae and Pythonidae. A retrovirus is suspected to be the cause of IBD, but has not been proven. Dr. Elliot Jacobson, from the University of Florida, College of Veterinary Medicine, is currently researching the disease and trying to determine if there is a relationship between the virus and formation of the cytoplasmic inclusions in infected snakes. IBD causes chronic regurgitation with quite variable neurologic disorders. During the early disease phase, boas may be asymptomatic or show signs of anorexia, weight loss, secondary bacterial infections, poor wound healing, dermal necrosis, and/or regurgitation. As the disease pro-

COMPANION ANIMAL, CONTINUED

gresses, neurologic signs may range from mild facial tics, abnormal tongue flicking, failure of the snake to right itself when placed in dorsal recumbency, to severe seizures. The course of the disease is more acute in pythons with neurologic symptoms being more profound. Severe neurologic disease is seen in most pythons and death usually occurs in days or weeks after the onset of clinical signs, unlike boas, where the active disease can linger for months before they become clinical.

Interestingly, two additional snakes from this same collection were diagnosed with IBD in February 2013. At that time, PCR testing was not available. Currently, the University of Florida is offering IBD PCR testing. Additional information regarding sample collection and submission may be found on their website.



Figure 1. Multifocal inflammatory nodules in the esophagus.

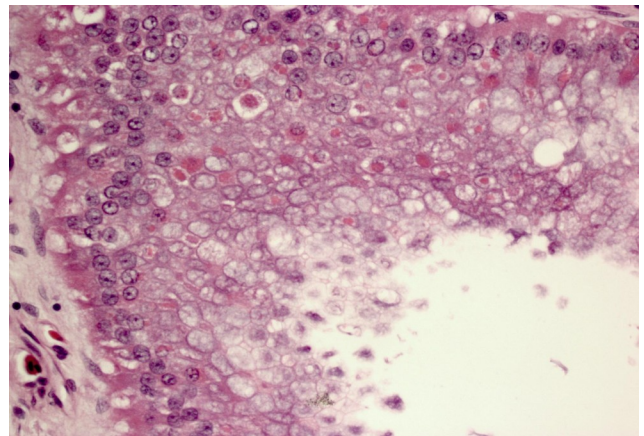


Figure 2. Eosinophilic intracytoplasmic inclusions in the renal epithelium. H&E, 40x

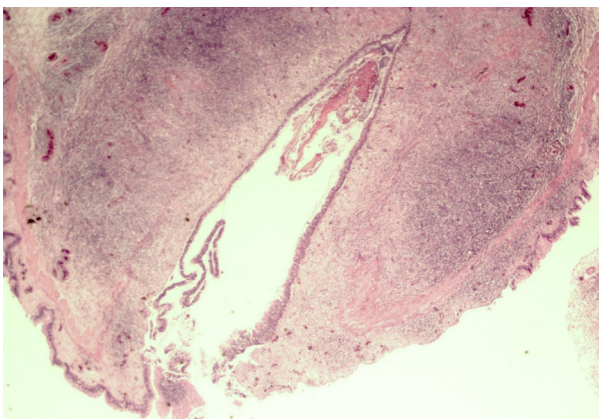


Figure 3. Inflammatory nodule in the gastrointestinal tract. H&E; 2x

COMPANION ANIMAL, CONTINUED

On November 19, 2013 an adult female Green Anaconda was seen by the attending veterinarian with a primary complaint of upper respiratory congestion and oral mucoid discharge. The snake held her head in marked extension and respiratory sounds were suggestive of upper respiratory congestion. Treatment with an injectable cephalosporin (Ceftazidime) was initiated. The snake subsequently died during the night of November 25. There are other snakes on the premises (number not specified) with none being reported as ill at the time of this submission. This particular snake reportedly had a case of pneumonia about 2 years previously.

Necropsy: The carcass is that of a 40.1 kg., adult female Green Anaconda in good nutritional condition and having mild post mortem change. A 10 X 8 X 7 cm firm, pale beige mass is situated just anterior to the base of the heart and is compressing the trachea. At the center of the mass is a 5 mm diameter cavity filled with dark, sanguinous fluid and containing a gray, irregularly spiculated structure. **(Figure 1)** The lung is light rose brown and resilient and a yellow-grey froth is noted in the airways. The liver is somewhat enlarged, friable, and with diffuse, pale mottling throughout. **(Figure 2)** Raised nodules of 5 mm to 1.5 cm diameter are distributed all along the esophageal mucosa. **(Figure 3)** Similar nodules are distributed along the colonic mucosa with many having crater-like ulcerations (fibrinohemorrhagic) at their surface and there is bloody colonic luminal content. The left thyroid is firm and white and measures 4 X 1.5 X 1.5 cm while the right is also firm and white but at 10 X 5 X 4 cm. A focus of organized hemorrhage (~ 7 mm diameter) associated with an ovary (presumptive) is loosely adhered to the hepatic surface.

Histopathology: **Lymphoma** present in lung, liver, kidney, intestine, stomach, trachea, ovary and oviduct. Additional tissue sections are composed entirely of sheets of neoplastic cells and contain no normal architecture, thus, tissue of origin cannot be determined. Neoplastic cells are present in the peripheral blood in multiple tissues (leukemia).



Figure 1: Mass located anterior to the heart base, and compressing the trachea



Figure 2: Sectioned liver revealing pale mottling of the parenchyma

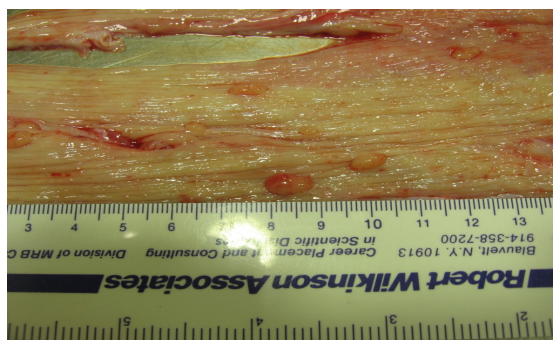


Figure 3: Raised nodules on the esophageal mucosa

LIVESTOCK

Cattle

A 75-day-old female Holstein calf was euthanized due to dehydration, lethargy, loss of body condition and fever. This animal and seven other calves were weaned and vaccinated for respiratory and clostridial disease about one week prior to illness. On gross examination, the rumen and reticulum were diffusely thickened with dense chunks of grain and short white hairs tightly adhered to the mucosa and multifocal to coalescing erosions and ulcers were evident. **(Figure 1)** Some sections of the rumen measured up to 7 mm in thickness. The rumen contained 3 liters of dehydrated grain with a pH of 6. Multifocal to coalescing erosions and ulcers were also identified in the omasum and abomasum. A necrotizing, fibrinous to fibrous, suppurative and hyperplastic rumenitis, reticulitis, omasitis, and abomasitis with intralesional bacterial and hyphal organisms were revealed on histopathology. Isolated from the abomasum were *Trueperella pyogenes*, *Pasteurella* sp. and *Rhizopus* sp.

This was a case of **chronic rumen acidosis (grain overload)**. The fungal invasion was due to the damaged rumen lining and destruction of rumen microflora. The submitter reported that a 90-day-old calf from the same group died and was diagnosed with a focal acute rumenitis, with rumen acidosis as the cause.



Figure 1: Chronic rumenitis

Mahogany Wade-Caesar, MS, DVM

Equine

The body of a 3 month old female Thoroughbred horse was presented to the Western Animal Disease Diagnostic Laboratory for post mortem examination. The provided history stated the horse had severe enteritis for the previous two weeks with signs of toxemia, dehydration, etc. The horse responded to intensive care, fluids and diarrhea therapy but would have cyclic fever despite intravenous antibiotics. Fecal culture was negative for *Salmonella*. Peritoneal tap results included 48,000 WBC/ml. Bacteria were present on tracheal wash. The referring veterinarian changed the antibiotics to Enrofloxacin and Metronidazole, but the horse still had a fever. The horse was taken in for exploratory surgery, where the referring veterinarian observed lymphadenopathy of the colon and cecum with adhesions.

The following gross lesions were seen during post mortem examination of the horse: Bilateral 4-5 cm white colored, firm masses in the medial lung lobes. **(Figure 1)** Multifocal areas of grey colored lung tis-

LIVESTOCK, CONTINUED

sue in the cranial lung fields. Irregularity shaped, marked enlargement of what appeared to be every lymph node associated with large colon and cecum. **(Figure 2)** The lymph nodes were firm and white colored on cross section. Also multiple bronchiolar and mediastinal lymph nodes were similarly enlarged. The majority of the small intestine had red discoloration of the intestinal walls and mucosa. The walls of the large intestine were thickened with the intestinal mucosa having a plicated appearance, white to red in color. The cecum contained grey to green colored fluid, and the large colon grey colored fluid contents.

Histopathologic lesions identified included: Severe, multifocal pyogranulomatous pneumonia and Chronic, severe, multifocal pyogranulomatous colitis. Acid Fast and Gram stains were negative.

Multiple tissues were submitted for bacterial culture. Cultures for *Salmonella*, *Clostridium perfringens* were negative, as were *Clostridium difficile* toxin assays test. Aerobic Culture of mesenteric lymph nodes was positive for 1 + growth of *Rhodococcus equi*.

The diagnosis was Pyogranulomatous colitis and pneumonia from a **chronic *Rhodococcus equi* infection**. The long term aggressive antimicrobial therapy that was continuing right up to the animal being euthanized was the likely reason *Rhodococcus* was not isolated for the other tissues cultured.

Except from the Merck Veterinary Manual concerning *Rhodococcus* stated "...Diarrhea is seen in one-third of foals with *R equi* pneumonia and may be caused by colonic microabscessation.... Intestinal and mesenteric abscesses are the most common extrapulmonary sites of infection. Foals with abdominal involvement often present with fever, depression, anorexia, weight loss, colic, and diarrhea. Intestinal lesions are characterized by multifocal, ulcerative enterocolitis and typhlitis involving Peyer's patches with granulomatous or suppurative inflammation of the mesenteric and/or colonic lymph nodes. The prognosis for foals with abdominal forms of *R. equi* is less favorable than for those with pulmonary disease."

Source: Merck Veterinary Manual, Online edition.



Figure 1: Pulmonary masses, medial lung lobe

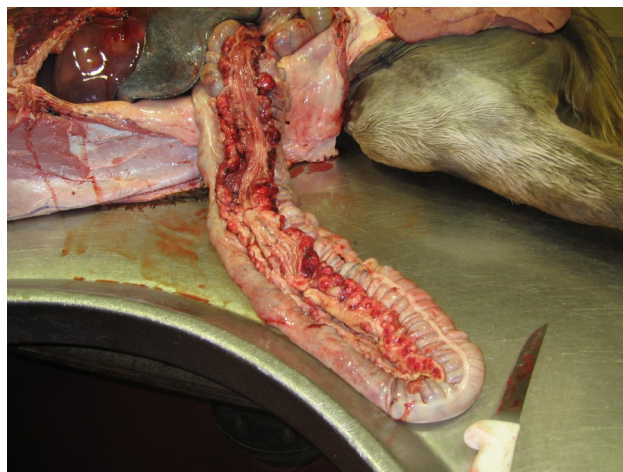


Figure 2: Large colon with associated enlarged mesenteric lymph nodes

LIVESTOCK, CONTINUED

An 18 year old Warm blood gelding was submitted to the Griffin Laboratory with a 2 day history of progressive anemia that was nonresponsive to treatment. The 540kg Warm blood gelding was submitted shortly after euthanasia. External exam revealed white mucous membranes. Ocular fluid was clear. Approximately 500-1000mls of hemorrhage tinged peritoneal effusion was noted. The diaphragm bulged into the abdomen in a convex shape. The thorax was distended with approximately 30L of dark red hemorrhage. The lungs were partially collapsed and ventral lung fields were dark pink and congested. Multiple 1-4cm diameter dark red foci were evident throughout the lungs lobes. Numerous (hundreds) dark red firm nodules ranging in size from 0.5cm-6cm diameter lined the pleura, **Figure 1**) diaphragm and covered the pericardial sac. (**Figure 2**)

Pathologic fractures were noted along ribs 7-8 on the left side mid rib and associated with the nodules. Multiple fragments were associated with those two ribs and the periosteum had a roughened texture. The myocardium was pale. The trachea was clear. An approximately 5-7cm hemorrhagic multi-nodular mass surrounded the left kidney and extended to the spleen. Nodules were noted along the spleen. The liver was pale. Histopathologic examination of the tissues was diagnostic for Hemangiosarcoma in the lung, kidney, spleen, pericardium and diaphragm.

Disseminated Hemangiosarcoma is reported to occur in mature, particularly middle-aged horses, with no apparent sex predilection. The respiratory and musculoskeletal systems were most commonly affected and presenting complaints included dyspnea, subcutaneous or muscular swelling, epistaxis, and lameness. Heart and respiratory rates were usually increased and mucous membrane color was frequently pale or icteric. Hemangiosarcoma should be included as a differential diagnosis for horses with evidence of hemorrhage into body cavities, skeletal muscle, or subcutaneous locations.

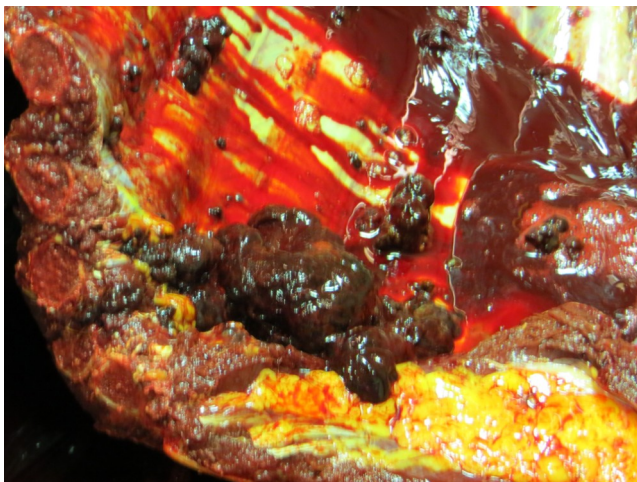


Figure 1: Hemangiosarcoma on the pleural surfaces



Figure 2: Hemangiosarcoma on the pericardial sac

LIVESTOCK, CONTINUED

Porcine

A 2.5-month-old Landrace pig was euthanized due to thumping (shallow, rapid breathing). On necropsy, the lungs failed to collapse upon entrance into the thoracic cavity, diffuse pulmonary edema was present, and the mediastinal lymph nodes were enlarged, reddened and edematous. The pericardial sac contained 60 ml of mildly turbid tan fluid. The mitral valve was partially effaced by a 5 x 3 x 2 cm yellow tan firm multinodular mass. The remainder of the mitral valve was thickened and nodular with petechial hemorrhages. **(Figure 1)** There was moderate dilation of the left atrium and ascites was present. Histopathologic examination revealed marked fibrinosuppurative and proliferative valvular endocarditis, myofiber degeneration and loss with interstitial fibrosis, and diffuse alveolar histiocytosis with pulmonary and pleural edema. Gross and histologic examination was consistent with **vegetative valvular endocarditis**.

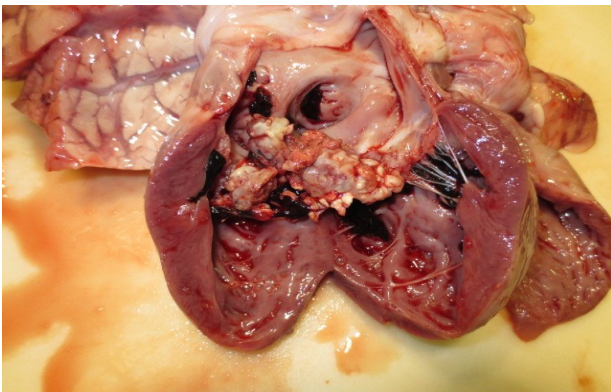


Figure 1: Vegetative valvular endocarditis of the mitral valve

Mahogany Wade-Caesar, MS, DVM

LIVESTOCK, CONTINUED

A 6 year old nulliparous Kune Kune sow was submitted with a history of anorexia and fever for approximately 2 weeks that did not respond to treatment. The sow weighed 60 kg and external exam revealed minimal autolysis and a tan vaginal discharge. The uterus was noticeably enlarged, weighed 5kg and filled most of the caudal abdomen. **(Figure 1)** Cut surface revealed cystic endometrial hyperplasia with variable sized clear fluid filled cysts covering the entire endometrium. **(Figure 2)** A small amount of yellow caseous material was noted in the left uterine horn. Histopathologic examination of the uterus confirmed cystic **endometrial hyperplasia (CEH)** and mild neutrophilic endometritis.

There is uncertainty in the literature as to whether the CEH is the result of progesterone or estrogen stimulation in pigs but the ovarian tissue in this sow would be more consistent with progesterone than estrogen. CEH is not an uncommon finding in adult pet pigs.



Figure 1: Gross, marked distention of the uterus

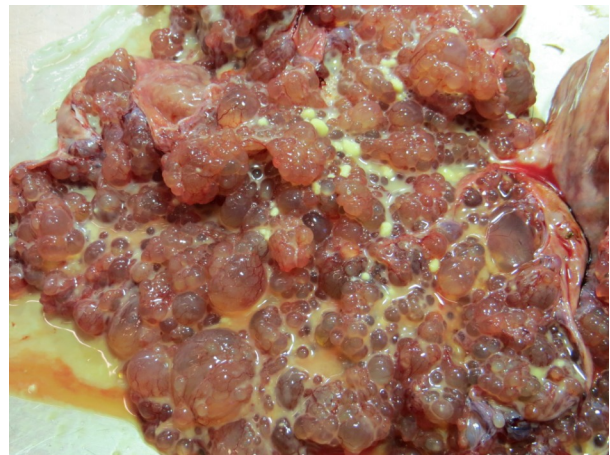


Figure 2: Multiple, various sized endometrial cysts

Kimberly Hagans, DVM

Wildlife

Two female Homing pigeons were recently submitted to the Griffin Animal Disease Laboratory. There had been weight loss, anorexia, diarrhea and weakness for about 2 weeks in some of the young birds in the flock. Yellowish exudate had been observed in the throat in some of the birds and Metronidazole had been administered for 1 week

Two deceased 2 month old, female birds were submitted. Both birds looked dehydrated and in good to slightly thin body condition.

Bird 1: in the oral mucosa there were areas of raised, tan to light yellow, necrotic exudate, congestion of the trachea, mild air sacculitis, dark red areas in the lung suggestive of pneumonia, mild swelling of the spleen, and watery, reddish brown intestinal content.

LIVESTOCK, CONTINUED

Bird 2: there were areas of raised, tan to light yellow, necrotic exudate in the mucosa of the esophagus (**Figure 1**) and to a mild degree in the caudal oral cavity.

Scrapings and direct microscopic examination of the oral cavity, esophagus and intestines were negative for parasites or ova.

Histopathology findings included basophilic intranuclear inclusions in the liver and spleen and vacuolar degeneration with necrotic debris in the oral cavity and esophagus.

Gross and histopathology lesions were consistent with **Pigeon Herpes Virus**.

Gross lesions seen in the oral cavity with Pigeon Herpes Virus can be confused with similar oral lesions seen with Trichomoniasis (Canker).

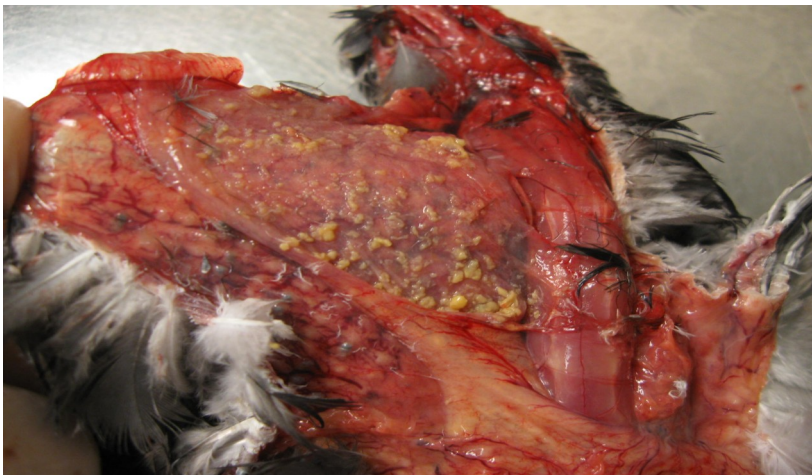


Figure 1: Pigeon Herpes Virus lesions in the esophagus

Reginald Ridenhour, DVM

LIVESTOCK, CONTINUED

On February 19, 2014 an opossum, found roadside and in "bad condition", was taken to the WNC Nature Center. Assessment suggested possible head trauma, e.g. eyes darting and flickering, unable to sit upright, constant shivering, (small) puncture wound of the left jaw, and treatment was initiated - antibiotic, corticosteroid, and subcutaneous fluids. Skin abrasions of the tail and toes were treated topically with Silver Sulfadine. It was also noted that there were numerous small white spots "all over the body" including both eyes. On the following morning the opossum was found dead with a milky white ocular discharge present.

Necropsy: The carcass is that of a 2.16 kg., adult male Virginia opossum in poor to marginal nutritional condition and having mild post mortem change. As indicated in the clinical history, there are superficial cutaneous abrasions of the tail and feet and one 5 mm diameter puncture of the skin just rostral to the left mandibular ramus that extends to the subcutis. **(Figure 1)** Myriad discrete white, firm, spherical nodules (1-2 mm diameter) are distributed throughout the skin with particular heavy densities noted on the lower, inner pinnae. **(Figure 2)** Nodules are present, as well, on and in the eye, **(Figure 3)** and the subcutis. **(Figure 4)** Much less numerous, but similar, nodules are scattered over parietal and visceral pleuroperitoneal and pericardial serous membranes. Scattered nodules are encountered within the parenchyma of some viscera to include lung, liver, kidney, spleen, heart, adrenal and testicle. Subtle purple-beige-dark brown splotching and mild to moderate firmness of texture are noted in the lung.

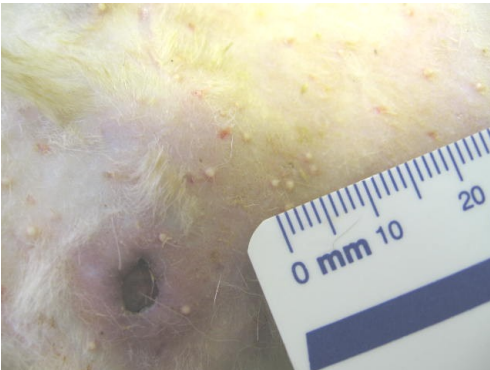


Figure 1: Skin puncture wound and cutaneous nodules



Figure 2: Nodules on the lower pinnae



Figure 3: Nodules on the corneal epithelium

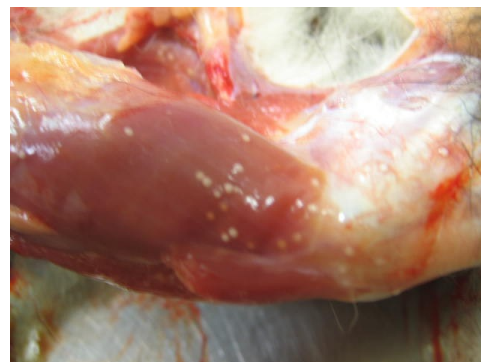


Figure 4: Nodules in the subcutaneous tissues

LIVESTOCK, CONTINUED

Histopathology: Large (up to 1 mm diameter), thick walled intracellular protozoal cysts containing myriad bradyzoites, consistent with *Besnoitia darling* were present within the heart, lung, kidney, lymph node, urinary bladder, urethra, dermis, choroid and iris. In addition, the tissues contained multifocal granulomas with mineralization consistent with ruptured cysts. - Alison R. Tucker, VMD, MA, Dipl. ACVP

Laboratory Diagnosis: *Besnoitia* Infection

Besnoitia infection in opossums is caused by the intracellular protozoan parasite *Besnoitia darlingi* of which the cat is the definitive host and, when eating infected opossum, is not harmed by zoites infecting and developing to oocysts in the intestinal epithelium. When shed oocysts are ingested by opossums (intermediate host) the parasites invade and form cysts within connective tissue components of almost any organ. Adverse effects due to *B. darlingi* infections in opossums are infrequent but the fact that the cysts are readily visible may prompt questions.

Richard Oliver, DVM

Ovine

An approximately 120 lb, female sheep in good body condition with moderate to marked postmortem autolysis was presented for necropsy with no indications of illness prior to death. The subcutaneous and visceral fat stores were yellow. The liver, lung, and kidneys were similarly dark red to black in color and the urinary bladder contained dark red urine. On histopathology, distal tubular red cell casts were noted in the kidney with occasional tubular necrosis and cholestasis was noted in the liver. The liver was submitted to the Pennsylvania Animal Diagnostic Laboratory System (PADLS) for a nutritional mineral screen. The copper level in the liver was 742ppm (ref. 25-100ppm) which confirmed a diagnosis of **copper toxicosis**. Sheep are the most susceptible species to copper toxicity and there are 2 forms that exist: 1) an acute form which involves ingestion of substances containing a large amount of copper and 2) a chronic form which involves ingestion of copper over a long period of time at doses below the acute toxic level. Potential sources of copper include: ingestion of feed and vitamin or mineral supplements designated for cattle, swine, or horses, ingestion of pasture fertilized with swine manure or poultry litter, and ingestion of copper-containing disinfectant foot baths for cattle. Certain plants (*Heliotropium europaeum* or *Senecio species*) that contain toxic alkaloids and are ingested over a period of time can cause sufficient liver damage and precipitate a sudden release of stored copper from the liver. Ingestion of other plants, such as the subterranean clover (*Trifolium subterraneum*) can produce a mineral imbalance resulting in excessive copper retention. Low levels of molybdenum or sulfate in the diet can also result in excessive copper retention.

Jessica Kaplan Kees, DVM

DEPARTMENTAL NEWS**ROLLINS LABORATORY****Departmental News/Rollins Lab New Hires**

Dr. Allison Boone, Anatomic Pathologist, June 2014

Clay Fox, Medical Laboratory Technician, Virology, May 2014

Richard Koch, Medical Laboratory Technologist, Bacteriology, March 2014

Summer Souther, Medical Laboratory Technician, Bacteriology, June 2014

ELKIN LABORATORY

Facility Update: Painting and new flooring in both large animal and poultry necropsy areas was completed June 18, 2014.

CE ATTENDANCE

Dr. Bradley L. Barlow gave a presentation to the Catawba Valley Cattleman's Association on March 11, 2014 about the North Carolina Veterinary Diagnostic Laboratory System, our services, and recent pertinent cases seen at the Northwestern Animal Disease Diagnostic Laboratory. Dr. Barlow also attended the Southeastern Veterinary Pathology Conference (SEVPAC) in Tifton, GA on May 17-18, 2014.

Dr. Mahogany Wade-Caesar attended the Central Veterinary Conference in National Harbor, MD on May 9-11, 2014.

Dr. Kimberly Hagans attended the 2014 Emerald Coast Veterinary Conference in Destin, FL during June 2014.

Dr. David Drum attended an Emergency Preparedness Training for Companion Animal and Livestock Emergency Issues conference in Greenville, SC on February 9, 2014.

Directory

Rollins Laboratory - 919-733-3986

Director

[Dr. Karen Post](#)

Assistant Director

[Dr. Richard Mock](#)

Veterinary Pathologists

[Dr. James Trybus](#) (Pathology Services Coordinator)

[Dr. Tahseen Abdul-Aziz](#) (Avian)

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Dr. Jennifer Haugland	Veterinary Diagnostician—NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Shannon Jennings	Corporate Poultry Practitioner—Nash Johnson Farms
Dr. Randy Jones	Private Veterinary Practitioner—Livestock Veterinary Services
Dr. Richard Kirkman	Private Veterinary Practitioner—Large Animal
Dr. David Marshall	State Veterinarian—NCDA&CS Veterinary Division
Dr. Karen Post	Director of Laboratories—NCDA&CS Veterinary Diagnostic Laboratory System
Dr. Rick Sharpton	Corporate Poultry Practitioner—Perdue, Inc.
Dr. Betsy Sigmon	Small Animal Practitioner—Creature Comforts Animal Hospital
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